

UNITED STATES DISTRICT COURT  
DISTRICT COURT OF NEVADA

LaKISHA NEAL-LOMAX, JOSHUA WILLIAM LOMAX, )  
ALIAYA TIERRAEE LOMAX, JUANITA CARR, as parent )  
and guardian of INIQUE ALAZYA LOMAX, and )  
JOYCE CHARLESTON, individually, and as Special )  
Administrator of the Estate of WILLIAM D. LOMAX, JR., )

Plaintiffs, )

vs. )

Case No.CV-S-05-01464-PMP-RJJ )

LAS VEGAS METROPOLITAN POLICE DEPARTMENT; )  
OFFICER REGGIE RADER, in his individual and official )  
capacity; SHERIFF BILL YOUNG, in his official capacity; )  
TASER INTERNATIONAL, INC., an Arizona Corporation; )  
TASER INTERNATIONAL, INC., a Delaware Foreign )  
Corporation; DOES I through X; DOES XI through XX; )  
and ROE CORPORATIONS XXI Through XXX, inclusive, )

Defendants. )

Expert Report: Vincent J.M. Di Maio, M.D.  
5 Reading Lane  
San Antonio, TX 78257  
Phone: 210-698-1400

Pursuant to Fed. R. Civ. P. 26(a)(2), I, Vincent J.M. Di Maio, hereby submit my report that contains a complete statement of all opinions to be expressed and the bases and reasons therefore; the data and other information I considered in forming the opinions; the exhibits or list of references I used as a summary of or support for the opinions; my qualifications, including a list of all publications authored within the preceding ten years; the compensation to be paid for the study and testimony; and a listing of any other cases in which I have testified as an expert at trial or by deposition within the preceding four years,

  
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Vincent J.M. Di Maio

April 19, 2007

\_\_\_\_\_  
Date



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April 16, 2007

As requested, I have reviewed the four books of material sent me in regard to the aforementioned case. Attached is the Index of the volumes.

William Lomax was a 26 year-old black male who, on the afternoon of 2/20/04, became involved in an altercation with housing authority security guards at the Emerald Gardens Apartment Complex in Las Vegas, Nevada. The guards approached him because of bizarre behavior manifested by irrational outbursts, impaired cognition and combativeness. There had been a similar encounter with Mr. Lomax on 12/17/03 due to his use of PCP (phencyclidine). At that time, he had been sent to University Medical Center where he admitted to use of PCP and demonstrated a sympathomimetic toxidrome manifested by tachycardia, hypertension and diaphoresis. As in the previous incident, the guards requested assistance from the police and medical personnel.

Officer Reggie Rader of the Las Vegas police department, who was at the complex for another call, saw the encounter between the guards and Mr. Lomax and went to their assistance. Officer Rader used his Taser in the stun mode in order to assist the guards in their efforts to handcuff Mr. Lomax. The Taser was applied to the base of the neck and activated approximately two times for 3 and 4 seconds, respectively. Two handcuffs attached end to end were then used to handcuff Mr. Lomax's hands behind his back. The witnesses at the scene stated that while use of the Taser would cause Mr. Lomax to cease his violent conduct, this reaction to its use was very transitory with rapid return to his aggressive behavior.

At approximately, 1758 hrs, a Las Vegas Fire Department engine arrived at the scene. The crew of four consisted of the senior officer, a firefighter, an EMT and a paramedic. The crew observed Mr. Lomax to be lying face down on the ground with his hands cuffed behind him with two pairs of cuffs. He was yelling, kicking and combative. Three guards and the police officer were attempting to hold Mr. Lomax down. The guard holding the right arm was applying pressure on Mr. Lomax's back with his knee. When subsequently ordered to stop this, he complied. Mr. Lomax continued to struggle and yell.

Shortly after the arrival of the engine unit, the AMR ambulance arrived. They made similar observations as the engine unit as to the condition of Mr. Lomax. The AMR personnel then removed a gurney from the unit and wheeled it to Mr. Lomax. Paramedic Ritz of AMR took out a Velcro restraint and attached it to Mr. Lomax's right arm. He gave Paramedic Pearson of the fire department a second restraint and the latter attached it to the left arm. Mr. Lomax was then

lifted onto the AMR gurney, and placed face down. At this time, he was agitated, yelling and combative. It was then decided to remove the cuffs and use soft restraints, i.e. the Velcro ties, to restrain him. During the struggle on the ground and then to put him on the gurney and replace the handcuffs with soft restraints Mr. Lomax was Tasered 5 times over a 2 minute period. The last two times, he was Tasered 2 and 6 seconds, respectively. After the last time, he became relatively docile and the cuffs were removed and he was tied to the gurney with the soft restraints. He offered resistance to placement of the Velcro wrist restraints according to Paramedic Ritz. A strap was also put across the legs. He was then assessed by paramedic Robert Pearson who determined he had a respiratory rate of 16 and a pulse of greater than 120 beats/m.

Mr. Lomax was then wheeled to the ambulance and placed on it. He was again assessed by paramedic Robert Pearson who determined he had a respiratory rate of 16 and a pulse of greater than 120 beats/m. A nasal cannula was inserted and an intravenous line started. He offered resistance to placement of the intra-venous line according to Paramedic Ritz. He was breathing when the nasal tube was inserted. He was then rolled over and found to be pulseless and apneic. This was a number of minutes after the last use of the Taser. An EKG showed asystole. Resuscitation was begun and he was transported to Valley Emergency Room arriving at 1850 hrs. The heart rate was restored but by this time he had anoxic encephalopathy. He subsequently developed acute renal failure, rhabdomyolysis and cardiac necrosis and was pronounced dead at 1325 hrs on 2/21/04. A toxicology screen was positive for PCP.

An autopsy was conducted at the Clark County Coroners Office on 2/22/04. The deceased weighted 233 lbs and was 70 inches tall. There were some minor external abrasions. Microscopic examination of the heart showed increased interstitial fibrosis and focal acute bronchpneumonia. Toxicological analysis of blood collected on 2/20/04 at 2000 hrs revealed a PCP level of 28 ng/ml while analysis of blood and liver collected at 1020 hrs on 2/22/04 revealed a PCP level of 129 ng/ml in the blood and 245 ng/mg in the liver.

Based on the aforementioned facts, it is my opinion that William Lomax died as the result of a fatal cardiac arrhythmia due to Excited Delirium Syndrome brought on by his use of the illegal drug PCP (phencyclidine). The increased interstitial fibrosis of the heart, which indicates prior injury to the heart, played a role in the death by making the deceased more susceptible to a cardiac arrhythmia. The presentation of asystole at the time of his cardio-pulmonary arrest rather than ventricular fibrillation is typical of deaths due to the Excited Delirium Syndrome. The microscopic pneumonia noted, in all medical probability, developed during his hospitalization secondary to his anoxic encephalopathy.

The use of the Taser did not cause this death. There is in fact no objective or scientific evidence that use of a Taser does cause death. The fact that he was restrained in the prone position also did not cause the death. Respiration in the prone position is more effective than in the supine position and use of restraints does not decrease the oxygenation of blood.

PCP was developed in the 1950s as an intravenous anesthetic. It is a noncompetitive antagonist of the N-methyl-D-aspartate (NMDA)/glutamate receptors. Use in humans was discontinued in

1965 because patients often became agitated, delusional, and irrational while recovering from its anesthetic effects. It is snorted, smoked, or ingested. The physical effects of its use include a rise in blood pressure and pulse rate, flushing, profuse sweating, and numbness of the extremities and loss of muscular coordination. Psychological effects mimic the symptoms of schizophrenia: delusions, hallucinations, paranoia, disordered thinking, and violent behavior. Repeated use of PCP may produce persistent symptoms of schizophrenia: psychosis, hallucinations, delusions, thought disorders, cognitive dysfunction and flattened affect. Symptoms can persist up to a year after stopping PCP use.

Excited Delirium Syndrome (EDS) involves the sudden death of an individual, during or following an episode of excited delirium, in which an autopsy fails to reveal evidence of sufficient trauma or natural disease to explain the death. In virtually all such cases, the episode of excited delirium is terminated by a violent struggle with police or medical personnel, and the use of physical restraint. In occasional cases, there may be use of Pepper Spray or a Taser in an attempt to control the individual.

Shortly following cessation of the struggle, the individual is noted to be in cardio-pulmonary arrest. Attempts at resuscitation are usually unsuccessful. If resuscitation is "successful", the individual is found to have suffered irreversible hypoxic encephalopathy and death occurs in a matter of days. Typically, the only findings at autopsy are minor abrasions and contusions explainable by the struggle that preceded death, as was true in this case.

The cause of death in cases of EDS is usually multifactorial, due primarily to a hyper-adrenergic state combined in some cases with the use of illegal stimulants, medications, natural disease and/or genetic polymorphism. In this case, death was due to activation of the Sympathetic System by the delirium, the struggle and use of PCP.

Deaths due to Excited Delirium were initially described in individual with mental disease, primarily schizophrenia and bipolar disease. The introduction of medication for mental disease in the early 1950's eliminated most such deaths. These deaths still occur in mental patients if the patient is off their medication. Most deaths of mental patients from EDS encountered by police are due to their stopping use of their medication and/or use of illegal stimulants.

Whenever one gets excited, such as in excited delirium, or engages in strenuous activity such as a struggle, or takes a hallucinogenic drug such as PCP, there is activation of the Sympathetic Nervous System with release of norepinephrine (NE) from nerve cells into the synaptic spaces between the Sympathetic neurons and receptor organs such as heart muscle and the coronary arteries. This causes the heart to beat harder and faster and to raise the blood pressure. At the same time, there is constriction of the coronary arteries with reduction of blood flow, and thus oxygen, to the myocardium.

Peak levels of catecholamines (norepinephrine and epinephrine) are reached not during the physical activity but in the 2-5 minutes after cessation of the activity and may reach 10x base levels. This is Dimsdale et al.'s "period of peril", when the heart is most sensitive to

development of fatal arrhythmias.

During the physical activity, blood potassium also increases. Elevated levels of catecholamines in the blood neutralize the arrhythmogenic potential of the elevated blood potassium. During the "period of peril", the blood potassium levels drop dramatically, at times to hypokalemic levels. Hypokalemia, like hyperkalemia, is arrhythmogenic, but its' effects are not protected by elevated blood catecholamine levels. Hypokalemia predisposes to prolongation of the QT-interval, development of *torsade de pointes* and sudden cardiac death.

Thus, anyone engaging in a struggle or strenuous activity, after cessation of the struggle, enters a "period of peril" characterized by peak levels of catecholamines and dramatically falling levels of potassium. While the usual result of these physiological changes is uneventful with a complete return to normal, in some individuals, especially those in excited delirium (ED), death can result.

In regard to the plaintiff's expert witness reports, a number of erroneous assumptions and conclusions are present. Thus, the mechanism of death is said to be respiratory arrest, though the facts disprove this. Mr. Lomax was observed to be breathing up to the time he arrested. For respiratory arrest to have caused the hypoxic encephalopathy, the brain would have had to been deprived of oxygen for 5-8 minutes. Thus Mr. Lomax would have had to have been in respiratory arrest for that period of time - which he was not. The effects of short term respiratory arrest would have been reversed by ventilation. The oxygen deprivation of the brain was due to cardiac arrest with lack of perfusion of the brain in spite of attempted cardiac resuscitation.

The experts contend that the prone position of Mr. Lomax with his hands cuffed behind his back caused hypoxia. In fact clinical research indicates that gas exchange is improved by the prone position and binding the hands behind the back when coupled with hogtying (a situation much more incapacitating than occurred in this case), produces only minor changes in ventilatory functions and, more importantly, no change in oxygenation of the blood.

The microscopic pneumonia noted at autopsy, in all medical probability, played no role in Mr. Lomax's cardio-pulmonary arrest but rather developed during his hospitalization secondary to his anoxic encephalopathy. Comments about neurological damage from the electrical current generated by the Taser are sheer speculation with absolutely no foundation. The references to permanent neurological damage in the literature refer to cases of electrocution.

Based on the aforementioned facts, it is my opinion that, in all medical probability, William Lomax died as the result of the Excited Delirium Syndrome with the excited delirium due to use of the illegal drug PCP. The mechanism of death was a hyperadrenergic state produced by the excited delirium, and the struggle. The increased interstitial fibrosis of the heart, which indicates prior heart injury, played a role in the death by making the deceased more susceptible to a cardiac arrhythmia. There is no scientific evidence that the use of the Taser caused or contributed to the death.

I am a physician Board Certified in Anatomical, Clinical and Forensic Pathology. Attached is my

Curriculum Vitae which gives details of my education, qualifications, professional experience and publications. I have testified in state and federal courts throughout the United States as well as in courts in Canada and South Africa. Also attached is a list of cases that I have testified in and a fee schedule. I reserve the right to amend this report should additional information be presented for my review.

Sincerely,

A handwritten signature in black ink, appearing to read "Vincent J.M. Di Maio". The signature is fluid and cursive, with a large initial "V" and "J".

VINCENT J.M. DI MAIO, M.D.